Passive smoking, potential atopy and asthma in the first five years

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Summary

Evidence of prolonged exposure to cigarette smoke was sought in a group of 86 children aged five years and under with moderately severe asthma, and in 1199 infants from a mixed background population of Armed Service and civilian families. Asthmatics with a normal serum IgE (less than +1 s.d. for age) made up almost half of the cases and, compared with those with an elevated serum IgE (+1 s.d. for age or more), a greater proportion were male, had experienced prolonged exposure to cigarette smoke, were from Service families and already had fixed chest deformity. It is suggested that, in addition to facilitating the expression of asthma in young potential atopics, passive smoking may be an important contributory cause of the more severe disease reported in the so-called 'intrinsic' group. Perhaps the burden of illness and the extent of exposure noted in this survey will prompt renewed efforts to be made to discourage smoking in families, particularly two years before and for at least five years after the birth of a child.

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Introduction

Passive parental smoking has been shown to be linked to respiratory infections, impaired lung development and bronchial lability during vulnerable periods in a child's growth and acquisition of immunity[1-4]. Despite the fact that passive smoking could well be the most important 'non-communicable' environmental factor involved in the aetiology of early asthma, only recently does it appear that a detailed account of this aspect of the child's early environment has been considered in prospective surveys of asthma following lower respiratory tract infections[5,6]. Even now, the extent to which passive smoking affects the severity and natural history of either atopic or non-atopic (perennial or 'intrinsic') asthma in small children is unclear.

Although the prevalence of parental smoking has shown a gradual decline in the past decade, the rate of fall is sex and social class related and there is evidence that the overall percentage of smokers and of smoking parents in South East Hampshire has generally been higher than in England and Wales as a whole[7]. There is also some evidence that parents of asthmatic children at age 15 years smoke less than the general population[8]. However, both at the Royal Naval Hospital, Haslar, and in the local community, the opposite was observed, i.e. the parents of very young children with a variety of obstructive airways diseases were often active smokers.

The aim of the present survey was therefore to define a population of young, more severe asthmatics and compare smoking behaviour in their parents with that of the general background population of parents locally in the Portsmouth area. At the same time, a more detailed assessment of atopic potential, immunization status, and chest deformity was undertaken in the asthmatic children.

Methods

Data on the background population were obtained by Gosport health visitors who enquired about regular smoking in both parents and other resident members of the household at the four-week examination for all children born in Gosport from April 1983 to March 1984 inclusive. This information was routinely collected as part of a multi-centre risk-related sudden infant death syndrome intervention study. An active smoker was defined as one who regularly smoked more than 5 cigarettes a day. A similar format of enquiry was used to ascertain those who had been resident in the household of asthmatic children and who had been active smokers for more than 50% of the child's first three years of life. An asthmatic was defined as any child under the age of six who had had three or more bouts of bronchitis or bronchiolitis (persisting cough and/or wheeze with illness lasting for 48 hours or more) in any six-month period and who in addition had had either definite intermittent wheeze or chronic night cough. This definition was used prospectively to screen all children referred to hospital with respiratory illness between April 1983 and January 1985, and the group thus comprised a consecutive series of young children with moderately severe asthma.

At the time of enrolment a careful enquiry was made by the author of family history of atopic symptoms in first-degree relatives, the number of previous admissions to hospital for chest problems and pertussis immunization status. An assessment was made of any chest deformity. Unless there was already clear and reproducible evidence of associated allergic eczema or urticaria, blood was taken for estimation of serum IgE and IgE antibodies to house-dust mite, grass, tree and weed pollens, and cat and dog (epithelium). Convalescent venous samples were taken when no oral steroid medication had recently been prescribed.

Atopic potential was thus assessed in three separate ways: in terms of the presence or absence of (1) a personal history of allergic eczema or urticaria; (2) a first-degree family history of atopic conditions; and according to (3) the level of a single convalescent estimation of serum IgE. Children regarded as having potentially non-atopic or 'intrinsic' asthma tended to show negative family histories, but, in particular, their serum IgE was less than 1 s.d. above the mean for age.

Results

In the 22-month period, 91 consecutively referred children aged under six years met the criteria for definition of asthma used for this survey. Comparison

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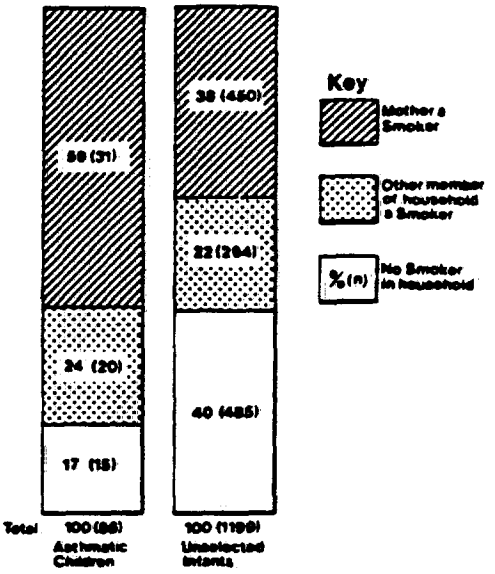

Figure 1. Prevalence of smoking in households of asthmatic children

with Ward diagnostic index and hospital statistics suggests that this group represented approximately one-quarter of all children seen with lower respiratory tract symptoms over the period and contained the majority of those with perennial and more severe illness. Five had persisting symptoms and signs and further investigations revealed specific diagnoses, namely: cystic fibrosis (1M, 1F), tracheo-oesophageal fistula (1M), congenital collapsing left main bronchus with hypoplastic left lung (1M), immotile cilia syndrome (1M). The remaining 86 (57M, 29F) had asthma with a mean age at ascertainment of two years and seven months, by which time they had already had a mean of 2.15 admissions per child. In 9 of these children the presence of atopic eczema, a positive family history of atopy in first-degree relatives, and reproducible allergic reactions or positive skin tests in the child strongly suggested atopy, and serum IgE measurement was not undertaken. This group was, however, included in the comparison of the prevalence of smoking in members of the household with that in the background population.

Prolonged exposure to cigarette smoke

The results of the comparison of prevalence of smoking in the households are shown in Figure 1. The prevalence of smoking was clearly higher in the households of asthmatic children than in the background population. When the differences in prevalence of smoking are broken down according to the father's occupation, as in Table 1, it is evident that the major difference between the Service and civilian populations lies in the greater proportion of members of households smoking in the Service asthmatic families (87% cf. 79%). Significance ($\chi^2_{(1)} = 11.0$, $P < 0.01$) for this group was the highest of the four sub-groups, all of which showed increased prevalence of smoking in the households of asthmatic children, compared to the background population.

Table 1. Prevalence of smoking in relation to father's occupation

	Armed Service		*Civilian*	
Mother a smoker:				
Asthma	23 (59%)	$\chi^2_{(1)} = 6.4$; $P < 0.05$	28 (60%)	$\chi^2_{(1)} = 7.8$; $P < 0.01$
Unselected	221 (37%)		229 (38%)	
Member of household a smoker:				
Asthma	34 (87%)	$\chi^2_{(1)} = 11.0$; $P < 0.01$	37 (79%)	$\chi^2_{(1)} = 5.6$; $P < 0.05$
Unselected	350 (59%)		364 (60%)	

Percentages refer to proportion of all service or all civilian

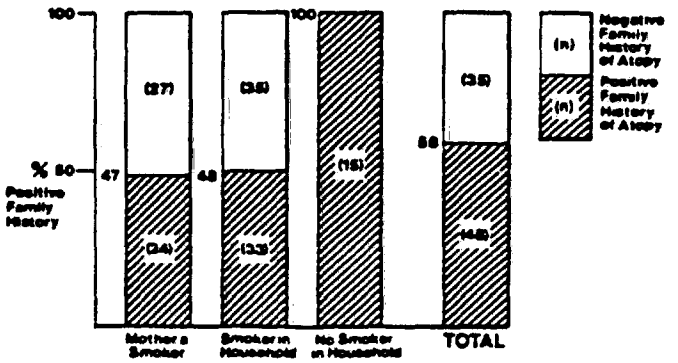

Figure 2. Smoking in households of asthmatic children in relation to family history of atopy

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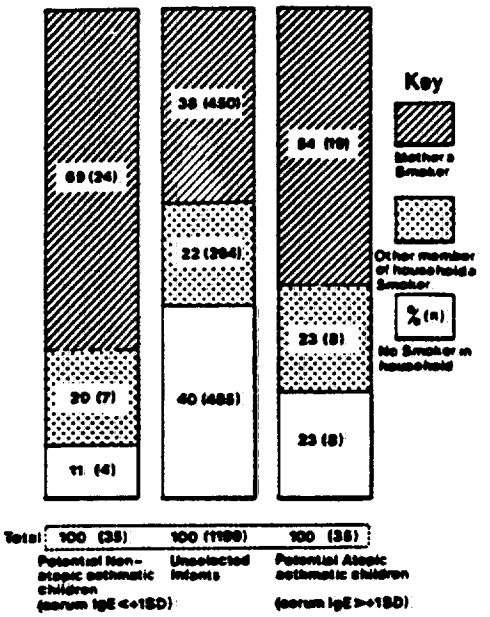

Figure 3. Prevalence of smoking in relation to atopic potential

Atopic potential

Family history: Information on family history of atopy in first-degree relatives was available in 83 of the 86 cases. From the data in Figure 2 it can be seen that in the absence of a positive family history, all the asthmatic children's households contained at least one member who was a regular smoker. All the non-smokers occurred in association with a positive family history of atopy, but even a personal or close family history of asthma did not appear to have discouraged smoking in the parents of many of these young asthmatic children.

Serum IgE: A single level of serum IgE was available for 70 of the 77 asthmatic children in whom atopic status was uncertain, but was unavailable in 7 either because they had moved from the area or defaulted the clinic.

Table 2. Pertussis immunization uptake for asthmatic children

	%	(n)
Potential non-atopic (serum IgE < +1 s.d.)	52	(27)
No smoker in family	50	(14)
Civilian family	46	(39)
Asthmatic children (all)	42	(69)
Member of household a smoker	39	(54)
Service family	37	(30)
Potential atopic (serum IgE > +1 s.d.)	35	(31)

The mean age of measurement of serum IgE in this survey was 2 years 11 months. On the basis of the single serum IgE measurement, 35 (50%) of the children tested were regarded as potentially atopic and an equal number potentially non-atopic. It can be seen from Figure 3 that a similar trend emerges, with the higher prevalence of active smoking occurring consistently in the households where the asthmatic child was potentially non-atopic. Differences between the potentially atopic and non-atopic households did not reach significance, but both of the groups with measured serum IgE showed significant differences when compared to the background population.

Pertussis immunization

For 69 of the 86 cases there was good recall of data on pertussis immunization status. The British Paediatric Association Immunization Committee's figures for pertussis vaccine uptake in the first three years of life (for 1982) are 53% for England and 59% for Wessex Region. Table 2 shows the comparison between the different sub-groups, with only 42% overall achieving positive status, i.e. in date with all scheduled immunizations. Thirty-three of this group of asthmatics (48%) had the double disadvantage of living in a household where there was an active smoker and being inadequately protected against pertussis.

Chest deformity

Seventy-one children were carefully examined by the author for evidence of fixed chest deformity, which in

Table 3. Chest deformity in asthmatic children

	With chest deformity Ratio	(n)	*Without chest deformity* Ratio	(n)	*Trends in those with chest deformity*
Family history of atopy					
Positive:negative	1.00:1	(30)	2.45:1	(38)	↓↓
Serum IgE					
Above +1 s.d.:below +1 s.d.	1.08:1	(26)	1.25:1	(36)	↓
Smoking in household					
Smoker:non-smoker	6.50:1	(30)	3.56:1	(41)	↑
Maternal smoking					
Smoker:non-smoker	2.00:1	(30)	0.96:1	(41)	↑↑
Pertussis immunization uptake					
Immunised:non-immunised	0.87:1	(28)	0.64:1	(36)	↑
Occupation of father					
Armed Service:civilian	1.30:1	(30)	0.46:1	(41)	↑↑
Sex distribution					
Male:female	2.29:1	(30)	1.56:1	(41)	↑↑

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most cases was manifest as a marked Harrison's sulcus and this was present in 30 (42%). More had developed chest deformity by the time of enrolment in the survey if they were male, came from Service families, had a negative family history of atopy, a serum IgE less than +1 s.d. (i.e. potentially non-atopic) and a mother or member of household who smoked (Table 3). Immunization against pertussis did not appear to confer protection against the development of chest deformity in this survey. These trends were not statistically significant. Overall a chest deformity was noted in 50% of those whose mothers smoked and 43% where a member of the household smoked.

Sex preponderance

Although there was a 66% male preponderance overall in the 86 cases (M:F 1.97:1), the preponderance in the group of potential non-atopic or 'intrinsic' asthmatics was 71% (25 of 35) compared to only 60% (21 of 35) in the potentially atopics; the male:female ratios were respectively 2.5:1 and 1.5:1. An increased male preponderance was seen also in asthmatics from Service families (2.8:1) and in those families where the mother smoked (2.1:1).

Discussion

Epidemiological studies of children with asthma and wheezy bronchitis have emphasized a high burden of illness, increasing prevalence and similar underlying mechanisms, with the need for a common approach to early diagnosis and management[8-10]. The recurrent ill health, multiple hospitalizations and early chest deformity seen in the young asthmatic children in this survey echo these findings. A most important variable, when comparing surveys of asthma, is difference in criteria used in definition[11]. Below the age of six, an exact definition of asthma is difficult to apply. The one used in this survey has proved helpful in focusing on early diagnosis and optimal management in both general practice and district paediatric unit settings.

The probability that at least two populations of young 'wheezers' might exist has been considered for some time, but it has not been clear whether these were allergic and non-allergic, atopic and 'intrinsic', or bronchitic and asthmatic[12,13]. The ventilatory response to exercise has been suggested as a good basis for separating such children. However, the poor reliability and reproducibility of bronchial lability testing in children under six years limits such a separation. Increased bronchial lability is considered to be a more likely explanation for the early male preponderance seen in young asthmatic children than atopic status which, in post-respiratory syncytial and other virus-induced wheezing, appears to be less important than it is in asthma in older children[14]. However, this explanation is not entirely convincing, and assessment of atopic status may have been inadequate in studies that have relied on family history alone[12].

Qualitative differences in the low levels of IgE at birth and in the first few years of life have permitted more accurate prediction of atopic respiratory disease, although in older children the relationship between single IgE levels and allergic symptoms remains controversial[9,15]. In Sweden, where some of this work has been carried out, the prevalence of asthma has been lower than in the UK and social conditions generally more favourable. It seems likely that some of the controversy which exists on the role of atopy has also arisen because variations in passive smoking and other important social factors have not been adequately taken into consideration in follow-up studies after virus, mycoplasma and bordetella infections, nor in bronchial lability studies on asthmatic children and their relatives[12,16]. In the most favourable social circumstances, however, atopy may more clearly be shown to predispose to asthma occurring with or as a sequel to infection[15-17].

The findings of the present Naval Hospital-based survey suggest that the majority (94%) of moderately severe young asthmatic children in whom atopic status is uncertain fall into one of three groups:

(*a*) Serum IgE > +1 s.d. above mean for age and member of household a smoker for > 50% of child's first three years (atopic, passive smoker).

(*b*) Serum IgE > +1 s.d. above mean and no smoker in household (atopic, non-smoker).

(*c*) Serum IgE within +1 s.d. of mean for age and member of household a smoker for > 50% of the child's first three years ('intrinsic', passive smoker).

A raised serum IgE and/or a mother who was an active smoker were noted in 84% of the 70 cases in whom IgE was measured.

It is particularly in the group of potentially non-atopic or 'intrinsic' asthmatics (Group C) that the major difference in male preponderance and parental smoking behaviour was observed. Exclusion of children with a personal history of eczema and a high local prevalence of smoking may have accounted for the high proportion of these 'intrinsic' cases (50% cf. 20% noted in other surveys)[13]. This separation into three groups may also help more satisfactorily to explain the early male preponderance in terms of the effects passive smoking might perhaps have in increasing bronchial lability and vulnerability to respiratory infections more in males than females.

The correlation of only 60.3% noted between raised serum IgE and positive family history of atopy in this survey might have been inferred from Kjellmann's findings[18] that although a family history of atopy was present in 50% of his cases with a similar atopic disease, the total incidence of atopic disease was only increased from 15% to 25% in those with a positive family history. The predictive value of serum IgE contrasts with the poor specificity of family history in providing a useful index of atopic status. In the present study, 5 children showed positive RAST tests, but serum IgE in the normal range, suggesting that there may be a significant group of mild atopics unascertained. It has been suggested that the effects of parental smoking on serum IgE levels in young children is to make the rate of rise with age more rapid and a significant difference has been shown at 36 months[19]. This would have had the opposite effect and made it more likely in the present study that atopic children would have been classified correctly.

Since the original survey undertaken by Colley *et al.*[1], there have been many others supporting their conclusions on passive smoking. The paper by Pullan *et al.*[5] in relation to respiratory syncytial virus infection is particularly clear in demonstrating the significance of maternal smoking, breast feeding, maternal care as assessed by the health visitor, and a single mother in relation to the severity of a young

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Figure 4. Paternal contribution - circa 1900. (Reproduced from Pipe Dreams, 1982, with kind permission of Pavilion Books)

child's respiratory illness. These findings are supported by the larger 'National Child Development' and 'Child Health and Education in the Seventies' studies in this country and numerous surveys from other parts of the world[2,3,17,20-24]. More recently Webb *et al.*[6], in a paper on continuing symptoms three and a half years after acute bronchiolitis, showed that of all the parameters (including family history of atopy and skin tests) considered, maternal smoking was the only one which, according to their data, reached significance at the 5% level. Other factors in addition to the development of the humoral response to house-dust mite and grass pollen antigens were inferred by Rowntree *et al.*[25] in their study on the continuing incidence of asthma at five years; and in a study on children at risk from atopic disease, Cogswell *et al.*[26] also noted that the one factor found to be associated with an increased prevalence of wheeze was the presence in the household of at least one parent who smoked.

Smoking behaviour may reflect a number of other social factors such as maternal stress. Medical care utilization is also closely bound up with parental smoking habit; nevertheless, a specific direct effect on aetiology of respiratory tract disease attributable to passive smoking seems likely. Some light may have been shed on the mechanism of this through the work of Tager *et al.*[4]. Acquired ciliary defects have been noted in nasal epithelia in children with respiratory infections, and it will be important to establish the frequency of similar defects in small children who experience significant passive smoking[27]. Cotinine estimations have established the existence of tertiary smoking and been helpful in illustrating quantitatively in the child the chemical effects of passive parental smoking[28,29].

Previous studies in Gosport have demonstrated that maternal smoking and other child care disadvantages assessed by health visitors were relevant factors in the prediction of infants at risk from sudden infant death syndrome in both the local Service and civilian populations[30]. The present survey illustrates that asthma and a family history of atopic disease are incorrectly regarded as contraindications to pertussis immunization. However, stress and adverse social factors are also suggested here and imaginative strategies will be required to counteract these[31]. The increased prevalence of parental smoking, poor uptake of pertussis immunization, and frequency of early chest deformity seen in Service families must be explained.

[redacted] Although health education programmes have not shown good immediate effect in general, a smoking cessation programme has been shown to be relatively effective in a group of sailors[32]. There remains scope for further programmes, and a clear statement is required of the necessity to avoid smoking in households two years before and at least five years after the birth of a child.

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References

1 Colley JRT, Holland WW, Corkhill RC. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. *Lancet* 1974;ii:1031-4
2 Fergusson DM, Horwood LJ, Shannon FT. Parental smoking and respiratory illness in infancy. *Arch Dis Child* 1980;55:358-61
3 Pedriera FA, Guandolo VL, Feroli EJ, *et al.* Involuntary smoking and incidence of respiratory illness during the first year of life. *Pediatrics* 1985;75:594-7
4 Tager B, Weiss ST, Münoz, *et al.* Longitudinal study of effects of maternal smoking on pulmonary function in children. *N Engl J Med* 1983;309:699-703
5 Pullan CR, Toms GL, Martin AJ, Gardner PS, Webb JK, Appleton DR. Breast feeding and respiratory syncytial virus infection. *Br Med J* 1980;281:1034-6
6 Webb MSC, Henry RL, Milner AD, Stokes GM, Swarbrick AS. Continuing respiratory problems three and a half years after acute viral bronchitis. *Arch Dis Child* 1985;60:1064-7
7 Nutbeam D. Wessex RHA, personal communication 1985
8 Tudor Hart J. Wheezing in young children: Problems of measurement and management. *J R Coll Gen Pract* 1986;36:78-81
9 Conway S, Littlewood JM. Admission to hospital with asthma. *Arch Dis Child* 1985;60:636-9
10 Levy M, Bell L. General practice audit of asthma in childhood. *Br Med J* 1984;288:1115-6
11 Godfrey S. What is Asthma? *Arch Dis Child* 1985;60:997-1000
12 Mok JYQ, Simpson H. Symptoms, atopy and bronchial reactivity after lower respiratory infection in infancy. *Arch Dis Child* 1984;59:299-305
13 Østergaard PA. Non-IgE mediated asthma in children. *Acta Pediatr Scand* 1985;74:714-9

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14 Verity C, Vanbeule B, Carswell F, Hughes AO. Bronchial lability and skin reactivity in siblings of asthmatic children. *Arch Dis Child* 1984;59:871–6
15 Kjellman NM. Predictive value of high IgE levels in children. *Acta Pediatr Scand* 1976;65:465–71
16 Hide DW, Guyer BM. Clinical manifestations of allergy related to breast- and cows'-milk feeding. *Paediatrics* 1985;76:973–5
17 Horwood LJ, Fergusson DM, Shannon FT. Social and familial factors in the development of early childhood asthma. *Paediatrics* 1985;75:859–68
18 Kjellman NM. Atopic disease in seven year old children. *Acta Pediatr Scand* 1977;66:465–71
19 Kjellman NM. Effect of parental smoking on IgE levels in children. *Lancet* 1981;i:983–4
20 Butler NR, Golding J. *From birth to five*. Oxford: Pergamon, 1986
21 Pengelly LD, Kerigan AT, Goldsmith CH, Inman EM. The Hamilton Study: distribution of factors confounding the relationship between air quality and respiratory health. *J Air Pollut Control Assoc* 1984;34:1039–43
22 Von Der Hardt H. Air pollution and bronchopulmonary disease in children (Germ). *Monatsschrift für Kinderheilkunde* 1985;133/1:2–5
23 Edwo EE, Weinberger MM, Lachenbruch PA, Huntley HH. Relationship of parental smoking and gas cooking to respiratory disease in children. *Chest* 1983;84:662–8
24 Tashkin DP, Clark VA, Simmons M, *et al*. The UCLA population studies of chronic obstructive respiratory disease VII: relationship between parental smoking and children's lung function. *Am Rev Respir Dis* 1984; 129(b):891–7
25 Rowntree S, Cogswell JT, Platts-Mills TAE, Mitchelle EB. Development of IgE and IgG antibodies to food and inhalant allergens in children at risk of allergic disease. *Arch Dis Child* 1985;60:727–35
26 Cogswell JT, Mitchell EB, Alexander J. Environmental factors and the development of allergic disease: effects of parental smoking, breast feeding and viral infections. Paper to British Paediatric Association, 1985
27 Carson JL, Collier AM, Hu SS. Acquired ciliary defects in nasal epithelium of children with acute viral upper respiratory infections. *N Engl J Med* 1985;312:463–8
28 Smith N, Austen J, Rolles CJ. Tertiary smoking by the fetus. *Lancet* 1982;i:1252
29 Jarvis MJ, Russell MAH, Feyerabend C, *et al*. Passive exposure to tobacco smoke: saliva cotinine concentrations in a representative population sample of non-smoking schoolchildren. *Br Med J* 1985;291:927–9
30 Powell J, Machin D, Kershaw CR. Unexpected sudden infant deaths in Gosport – some comparisons between service and civilian families. *J R Nav Med Serv* 1983;69: 141–50
31 Nicoll A. Contraindications to whooping cough immunization – myths or realities. *Lancet* 1985;i:679–80
32 Soul JO. Smoking cessation, experience at sea using nicotine chewing gum and including a period of active service. *J R Nav Med Serv* 1984;70:91–3

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